HAEMATOMYELIA.

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It is a striking fact that while we so often meet with hemorrhage into the brain, hemorrhage into the spinal cord, not produced by any trauma, belongs to the rarest affections known in neuro-pathology. It has indeed been denied that we can ever speak of a primary spinal hemorrhage, the idea being that a softening of the tissue of the cord must always precede, and that the hemorrhage is thus only a secondary one. Monod, as early as 1830, doubted its existence; but the chief exponents of this view are Charcot, and after him Hayem.[1] This latter author has collected the cases, which up to his time were reported under the title of Haematomyelia, and has subjected them to a careful analysis. Among the thirty-one cases there was not one, Hayem thinks, in which we could reject the supposition that a change had taken place previous to the hemorrhage and so altered the tissue of the cord as to produce an extravasation of blood. Such a position, however, has been taken only by the just mentioned authors, while all the later ones, as, *e. g.*, Leyden, Eichhorst, Gowers, Erb, admit the existence of primary haematomyelia.

Previous to Hayem, Levier[2] in his monograph had given the histories of sixteen cases, fourteen of which are included in the thesis of Hayem. Since Hayem's time eleven more cases have been described, a collection of which we find in Berkley's paper on Syringomyelia.[3] I have been unable to find any further one reported with autopsy.

It would necessitate the writing of a monograph to review again all the cases and to discuss their exact nature, and it would in fact be an impossible task for, as Leyden[4] and Eichhorst[5] already have

[1] Hayem: Des hémorrhagies intrarachidiennes. Thèse des Paris, 1872.

[2] Levier: Beitrag zur Pathologie der Rückenmarksapoplexie. Inaugural Dissertation, Bern, 1864; also, Schweizerische Zeitschrift für Heilkunde, 1864, Band 3.

[3] Brain, 1889.

[4] Leyden: Ein Fall von Haematomyelie. Zeitschrift für Klin. Medicin, Band XIII, 1887, pp. 225–251.

[5] Eichhorst: Beiträge zur Lehre von der Apoplexia in der Rückenmarkssubstanz. Charité Annalen, Berlin, 1876, pp. 192–205.

pointed out, we may be unable, even with a microscopical examination, to settle the question whether we have to deal with a primary softening and a secondary hemorrhage or with a primary hemorrhage and a secondary softening, analogous to the condition in the brain. Eichhorst and Gowers, therefore, admit, in opposition to Hayem, the possibility that some cases described as myelitis are really haematomyelia.

We see, therefore, that an anatomical diagnosis is not always possible, and that the clinical history with its different factors will be of great importance.

Undoubtedly, however, not a few cases described as such are not true examples of primary haematomyelia, but on the other hand cases like that of Goltdammer[1] *e. g.*, cannot be interpreted in any other way. Recently two cases have been reported by Krafft-Ebing,[2] diagnosed *intra vitam*, which we have every reason to call primary hemorrhage, also two cases by Sonnemann[3] from the clinic of Mendel.

It has been found by Charcot and Bouchard, and it is now a well-known fact, that the existence of miliary aneurisms in the cerebral arteries is a very common occurrence, so common that the above mentioned observers found them in seventy-seven consecutive cases of cerebral hemorrhage which they examined. This is, therefore, the most common cause of cerebral hemorrhage. We know that we find arterio-sclerosis in the spinal as well as in the cerebral arteries, but the soft pulpy brain with its comparatively large arteries and a high arterial pressure must be a more favorable situation for the formation of aneurisms than the denser spinal cord with smaller arteries and lower pressure,—not only for the formation of aneurisms but also for a hemorrhage to take place, sufficient to produce perceptible symptoms. Small hemorrhages are not unfrequently seen. But there are cases in which aneurisms similar to those in the brain have been described. Griesinger[4] reported a case where he found aneurismal

[1] Goltdammer: Beiträge zur Lehre der Spinal-apoplexie. Virch. Arch., Bd. LXVI, 1876.

[2] Krafft-Ebing: Zur Kenntniss der primären Rückenmarksblutung. Wiener Klinische Wochenschr., 1890, p. 939.

[3] Sonnemann: 2 Fälle von Haematomyelie. Inaugural Dissertation, Berlin, 1890.

[4] Griesinger: Archiv. für Heilkunde, Band 3, 1862, p. 174. Quot. by Levier (*loc. cit.*).

dilatations in the cord of a boy who had died with tetanus, Hebold[1] in a case in which he attributed them to obstruction from venous thrombosis. Of special interest is the observation of Eichhorst,[2] who found in the case which he described as primary haematomyelia quite wide-spread aneurisms,—furthermore, the case of Wilkins[3] whose patient died with an acute spinal paralysis, regarded by the author as haematomyelitis, and where the existence of aneurismal dilatations in the grey matter of the upper dorsal region was noted.

While in the cases which we shall describe in this article we cannot entirely exclude the trauma, still we cannot class them under the head of the usual traumatic cases in which injury or concussion to the spine was immediately followed by a hemorrhage, since in one case six days, in the other three weeks, intervened between the accident and the paralysis.

The cases, therefore, seem to be of sufficient interest to be reported.

I am indebted to Professor Osler and Dr. Thomas, the chief of the Neurological Clinic, for the permission to report these cases. Case No. II has been reported by Dr. Thomas as "A case of Brown-Séquard's paralysis" before the medical society of the Johns Hopkins Hospital.

Case I.—K. I., aet. 40, an Irishman, laborer, came to the out-patient department on July 26th complaining of partial loss of power in his right arm and hand.

The account of the family history of the man is somewhat incomplete, since he has for many years not heard from his people. In previous years, and up to his present illness, he has had unusually good health and has not lost a day's work in the past fifteen years. He denies very positively any venereal infection, and says that he has for the past six years been a very moderate drinker, while previous to that he drank too much at times.

In the first week of June, 1890, while at work he was struck between his shoulders by a board which fell from the height of eight

[1] Hebold, Otto: Aneurismata der kleinsten Rückenmarksgefässe. Arch. f. Psych., Bd. 16, 1885, p. 813.

[2] Eichhorst, *loc. cit.*

[3] Wilkins: Case of spinal apoplexy. Montreal General Hospital Reports, Vol. 1, 1880, p. 111.

or ten feet. He was knocked down but immediately got up again and felt no bad effects from the blow except some soreness on the following day at the spot where the board had struck. He went to work and felt well in every way up to about three weeks after this had happened. He came home one evening after a usual day's work, took his supper and felt quite well. He went on the street and sat down talking to a neighbor, only about half a square from his dwelling, when he was suddenly seized with a stabbing pain between his shoulders. He immediately arose and walked home, which he could do without assistance, noticing nothing at all in his legs. Soon he felt pain down the back, and when he lay down suffered from quite intense pain which had now also spread to both arms. The arms were drawn up across his chest and were tremulous. He remained in this condition for a few minutes and then completely lost power in his legs and partly in his arms, chiefly the right.

The time which elapsed between the onset of the pain and the paralysis was not more than eight or ten minutes. He did not lose consciousness at any time. Both arms could be moved at the shoulder-joints to some extent; the left was less affected than the right. He did not feel the people who came and rubbed him, nor could he feel a mustard plaster which was put upon his abdomen. He thinks he lost sensation completely on the body from about a line with the nipples down and in the arms. The physician ordered him a "sleeping powder," which had the desired effect, and the next morning he had no pain but felt numb in the places where the pain had been. His left leg could be moved a little and got better rapidly. About five days later he began to move the toes of the right foot, but the right leg improved more gradually than the left. Three days after the attack he could not hold anything in his hands. Both arms improved, the left more quickly and much more completely, the improvement in the right being chiefly in sensation. In general, sensation returned with motion. He could not pass his water for about two weeks, and the catheter had to be used. For some days also he had no stool, and took about the fourth day a purgative which acted. There was no priapism. As stated above we did not see the patient until July 26th, *i. e.*, about a month after the paralysis had come on. His condition then was as follows:

Status Praesens.—Rather tall man. Face thin. Expression anxious. Muscles fairly well developed, little adiposa. Intelligence good. No abnormality about voice. Eye-balls freely movable. Left pupil larger than right, both react to light and accommodation. No trouble with vision, and in general with none of the special senses. Muscles of face act equally. Motions of head executed normally and with normal strength.

No tenderness and nothing abnormal about the spine. Trapezius (occipital portion) is more prominent on the right than on the left side. Shrugs shoulders and resists depression. Deltoids act on both sides and in all directions. Pectoralis somewhat subnormal on the right side. Rotates arm. Flexors of forearm act on both sides, somewhat weaker on the right. Extensors on the right very weak, on left strong. Motion about wrist on the right side is limited to the slightest flexion and extension. No motion in fingers. No contracture. On left side all motions of hand and fingers are possible, power is very subnormal. Muscles react to mechanical stimulation and upon being percussed a welt is formed at the place of contact.

I shall here only give an extract of the electrical examination, which was then not so complete as a later one, which will follow in extenso. In the left arm there was no electrical change found to faradism; with the galvanic current the muscles and nerves could be excited with about 2 M. A., K. C. C. with a corresponding A. C. C. In the right arm with the faradic the deltoid, biceps, triceps, supinat. longus, extens. radial., act well at about 41., as do the pector. and triceps—the triceps less than the others. Flexors do not act. Radial and ulnar nerve not obtained. With the galvanic the radialis, supinat. longus, extensor radial., biceps, deltoid, reacted quickly to a stimulation with 3 M. A., K. C. The triceps reacted less readily and rather sluggishly, and the A. C. C. was even somewhat larger than the K. C. C. The extensor communis, extensor ulnaris, extens. pollic., extens. indicis, showed slow contractions of a tetanoid character. The anode, however, being larger than the kathode. The flexors were excitable but reacted slowly; there were no qualitative changes in the reaction.

Roughly tested no sensory change was found. Heart and lungs were normal. The patient then left and did not return until Janu-

ary 31st, when he was again examined, and the following notes were made:

Both legs look and measure equal. On a more careful examination no change is found in sensation to touch, temperature or pain, and faradism. Walks well. Pateller reflexes are active and equal on both sides, cremast. and plantar present. Electrically, all muscles and nerves of legs react well to galvanic and faradic, no diminution and no qualitative changes. Muscular strength good throughout in legs.

Body and arms: The right mammary region looks somewhat flattened, so that the intercostal spaces are more visible especially towards the sternum. The back looks symmetrical. Deltoids equal, and the muscular strength good in them. There is only slight difference in the pectoral. in favor of the left. The right upper arm is considerably thinner, and at the triceps seems especially flabby. Forearm on the right side is also thinner. The flexors especially seem flabby here. The right hand looks somewhat glossy. The surface over the knuckles and on the fingers more even than in a normal hand. The fingers are slightly bent, the hand extended. There is distinct atrophy of the thenar eminence. The left hand looks natural, except that the thenar of this side looks also flattened.

Measurements.	*Right.*	*Left.*
Wrist	14.8 cm.	16.0 cm.
Over supinat., with arm flexed at right angle	22.5 cm.	24.0 cm.
Greatest circumf. of upper arm	12.5 cm.	26.0 cm.
With arm extend. 13 cm., above ex. malleolus	19.0 cm.	21.0 cm.

Muscular strength of bicipites is fair and almost equal. Triceps is normal on the left side, very much subnormal on the right. The right hand can be flexed dorsally, but is held to the inside (radialis extensor). The left hand is raised higher and in median line, and the muscular strength in these muscles is good. Hardly any palmar flexion is possible in the right hand, while it is good in the left hand. No motion is possible in the fingers of the right hand; the thumb can be slightly separated from the other fingers. The motion in the fingers and hand of the left side is complete.

Electrical Examination.—We would state here that the higher the number is, which is given as the point of the faradic stimulation, the stronger is the current, for the scale of our induction coils is arranged relatively, 100 being the strongest current.[1]

A medium-sized electrode was put on the sternum and for the different [Erb] electrode a button-electrode was used, and the smallest traction noted.

Faradic Current.

	RIGHT.	LEFT.
Biceps	13	17–22.
Deltoid, anterior part	17–22.	17–22.
Deltoid, posterior part	17–22.	
Median nerve	28 (Pronat.)	28.
Ulnar nerve	Not obtained.	28.
Radial nerve	Not obtained.	22.
Triceps	17–22, slight and sluggish	22–28.
Supinator longus	17, good motion.	17–22.
Radialis extensor	28, good motion.	
Ulnaris extensor	Not obtained.	
Extensor communis	Not obtained.	
Interossei	Not obtained.	
Thenar	Not obtained	41–46, sluggish.
Hypothenar	Not obtained.	Adduct. pollicis, 41–46.
Other small muscles	Not obtained.	
Flexors	Not obtained.	
Pronator	22.	

Pectoralis reacts less on right than left, the difference is mainly in the lower part of the pectoral muscle.

Galvanic.

Right Arm.—

Biceps, 2 M. A., K. C. C. < 3 M. A., A. C. C.
Deltoid, anterior part, 3 M. A., K. C. C., 3 M. A., A. C. C. On stronger current, K. C. C. > A. C. C.
Lateral part, 1.5 M. A., K. C. C. < 4.5 M. A., A. C. C.
Posterior part, 2.5 M. A., K. C. C. < 3 M. A., A. C. C.
Triceps, 3 M. A., K. C. C. < 5 M. A., A. C. C.
Supinator, 2.5 M. A., K. C. C. < 4 M. A., A. C. C.
Ulnaris extensor, 2.5 M. A., K. C. C. sluggish < 3.5 A. C. C. sluggish.
Extensor communis, 4 M. A., K. C. C. slow, 4 M. A., A. C. C. somewhat quicker.

[1] The apparatus is described by Dr. Thomas in the *Bulletin* of the Hospital, July, 1890.

Thenar, 1½ M. A., K. C. C. slow. < 3 M. A., A. C. C. slow.
Adductor pollicis, 1 M. A., K. C. C. slow. < 3 M. A., A. C. C. slow.
Opponens pollicis, 1 M. A., K. C. C, slow. < 2 M. A., A. C. C. slow.
Flexor profundus, 2 M. A., K. C. C. < 3 M. A., A. C. C. very slow.
Sublimus, 3 M. A., K. C. C. < 5 M. A., A. C. C. very slow.
Ulnar and radial nerve not obtained.

Left Arm.—

Radial nerve, 2.5 M. A., K. C. C. < 4.0 M. A., A. C. C.
Biceps, 1 M. A., K. C. C. < 3.5 M. A., A. C. C.
Triceps, 2 M. A., K. C. C. < 4 M. A., A. C. C.
Ulnar nerve, 1.5 M. A., K. C. C. < 2.5 M. A., A. C. C., 2.5 M. A., A. O. C.
Supinator, 1.5 M. A., K. C. C. < 2.5 M. A., A. C. C.
Radialis extensor, 2 M. A., K. C. C. < 3 M. A., A. C. C.
Extensor communis, 2 M. A. K., C. C. < 3.5 M. A., A. C. C.
Extensor ulnaris, 2.5 M. A., K. C. C < 3.0 M. A., A. C. C.
Adductor pollicis, 2 M. A., K. C. C. < 4.5 M. A., A. C. C.
Extensor minimi digiti, 1 M. A., K. C. C. < 2.0 M. A., A. C. C.
Interrossei, 2 M. A., K. C. C. < 3 M. A., A. C. C.
Adductor pollicis, 2 M. A., K. C. C. < 2.5 M. A., A. C. C. rather slow.
Thenar, 1.5 M. A., K. C. C. < 2.0 M. A., A. C. C.

As regards the diagnosis of this case we have anticipated it in the title of this article. But if authorities like Leyden have expressed their opinion as to the impossibility of diagnosing *intra vitam* a haematomyelia it seems only natural that the reasons should be given why this seems the most probable diagnosis. A short recapitulation of the case will, therefore, I think, not be found out of place.

Patient was struck between his shoulders by a board falling from the height of eight to ten feet without producing any immediate effects save some soreness next day, which soon passed off and left him quite well. Three weeks later he was suddenly seized with acute pain between the shoulders radiating down the spine and into the arms, followed by a contracture of short duration in the arms, and within eight to ten minutes by complete sensory and motor paralysis from the arms down (with the exception of the higher-situated muscles of the arm). Retentio urinae et alvi. Improvement in the left leg the following day, comparatively rapid improvement also in the right leg and left arm, so that after three weeks there remained only paralysis of some of the muscles of the right arm, especially the forearm. On examination a month after, no sensory changes but paralysis with atrophy, showing the characteristic electrical changes of certain muscles of his right arm.

We have, therefore, to deal with a sudden spinal paralysis which has destroyed the grey matter in a certain region of the cord. Before going into the question to what the lesion is due, let us localize it as far as it is possible, by finding out which muscles—as indicated by their impairment in motion and visible atrophy and by the results of the electrical examination—are interfered with in their nutrition, and we can then draw conclusions as to their trophic centres in the cord.

The muscles concerned are the following, classing them under two heads, namely, those which have completely lost their motion and show reaction of degeneration, and those which show only decrease in electrical excitability and some impairment in motion.

It will be seen, however, in the tables of the electrical examination that even those muscles which are the most degenerated do not fulfill all the requirements of the reaction of degeneration, as it has been given by Erb, but that we find the anode-closing contraction nowhere larger than the kathode-closing contraction, a condition which is not rarely seen.

I. *Muscles showing complete loss of motion and reaction of degeneration.* Extensor ulnaris, extensor communis; all the flexors; small muscles of the hand.

II. *Muscles showing decrease in electrical excitability and impairment of motion.* Lowest part of pect. major and triceps.

Now on what basis can we make an attempt to localize the lesion? A very important contribution to the spinal localization has been given in the valuable work of Thorburn.[1] From results of purely clinical observation he has mapped out a scheme of the distribution of the motor-nuclei, to which much importance is to be attributed, as the conclusions were drawn from a sufficient number of cases, observed and studied with care; and since they coincide in general with the anatomical investigations of Herringham[2] and the stimulation experiments of Yeo and Ferrier.[3]

According to Thorburn we have to place the lesion on a level with the VII and VIII cervical and I dorsal roots and partly upward in the VI cervical, and this as we will see seems to be the most plausible.

Thorburn's scheme is as follows:

[1] A Contribution to the Surgery of the Spinal Cord, by Wm. Thorburn. Philadelphia, Blakiston.

[2] Proceedings Royal Society, No. CCXLIII, 1886, p. 255. Quoted by Thorburn.

[3] Proceedings Royal Society, No. CCXII, p. 12. Quoted by Thorburn.

VI. Subscap. pronat. teres. maj.; lateral dorsi. pectoralis maj.; triceps, serratus magn. VII. Extensor of wrist. VIII. Flexor of wrist. I. Dors: Interossei and other intrinsic muscles of hand.

The small muscles of the hand are by the three mentioned authors placed lowest down in the cord, the flexors and extensors in the region of the VII and VIII, by Ferrier, also in VI, V, IV, by Herringham in VI. The pectoralis could be stimulated by Ferrier from the VII and VIII, Herringham could trace fibres into VI, VII and VIII, while Thorburn refers its centre to the VI. It seems quite likely that the pectoralis has its centre in more than one segment of the cord. Both our cases, the one just reported and the one yet to follow, seem to point to this, for in both the lower part only was diseased, and we would conclude then that the centre for the lower part is situated lower (VI), while that for the upper is placed higher (V).

Another interesting point as regards localization is that we find the radialis extensor entirely preserved in our case. Thorburn does not speak of any nucleus for this muscle on a separate level from the others, while we find it especially mentioned by Herringham (VI and VII cervical). It is most probable that this muscle is represented higher up in the cord than the other extensors, for we have evidence of complete destruction of the anterior grey horns at the place where they are represented, while the extensor radialis remains quite intact. We see then that in general, according to the localization given by Thorburn, the muscles which we find diseased are represented in the cord in close connection, so that we can well understand how a hemorrhage in the grey substance, extending over somewhat more than three levels, can destroy their nuclei. So much then for localization.

Before going into the differential diagnosis, I propose to give the notes of the second case.

CASE II.—L. I., aged 21, railway brakeman, came to the surgical ward of the Hospital complaining of trouble in his hip, which proved to be a haematoma of considerable size. He was operated upon for this and left the Hospital well, as regards his surgical trouble, in the middle of January, 1891.

On October 18th, 1890, he was sent to the neurological clinic for examination because he complained of not being able to move his whole right side freely, the leg especially being somewhat stiff; he had also some disturbance in sensation in the left side.

Anamnesis.—Patient had always been healthy and strong. Sometimes he has been drinking rather heavily. He denies any venereal infection. On the 18th of July patient met with an accident. While standing on a freight car he was knocked down and fell under the cars in the middle of the track, so that he was somewhat injured by two other box cars passing over him. He did not lose consciousness. The only wounds he had were on the forehead, and at different places on the scalp, and also on left foot, but his spine was uninjured and there was no sign of paralysis.

Six days after—he had meanwhile been brought to a hospital—he felt some pain in his right arm and leg, and did not sleep very much that night; when he awoke in the morning the right side, with the exception of the face, was completely powerless. His speech was not impaired. He slowly improved, the first sign of improvement being in the deltoid, then in the leg, while the fingers for some time were in a semi-flexed position, so that he neither could flex nor extend them entirely. Soon after the paralysis of motion he noticed also that on the sound side he could not feel as well when pinched as he could on the other. There was retention of urine and faeces for a few days and he had to be catheterized once.

Status Praesens.—December 12th. Strong-looking man. Walks with difficulty, on account of the haematoma on the left hip and apparently some weakness in the right leg. The hip is bandaged. Facial muscles act well and equally. Eyes are freely movable, normal. There is no abnormality seen on the spine and no tenderness. Shoulders look equal; the lower part of the right pectoral is somewhat flattened, as compared with the left. The triceps feels more flabby on the right side; the upper and lower arm somewhat thinner. The arm and hand held in natural position. The interosseal spaces are more marked on the right hand. The thenar eminence is quite flattened. Every motion seems possible.

Measurements.—	*Right.*	*Left.*
Upper arm, largest circumference	28½ cm.	29¼ cm.
Fore arm, largest circumference	28½ cm.	31½ cm.
Wrist	20¼ cm.	20½ cm.

Muscular Strength.—Deltoids good and equal. Pectorales: The left pectoral muscles are stronger. On contracting them the lower

part of right pectoralis major is seen to be distinctly wasted. Extensors of the forearm: good on the left, much subnormal on the right. Flexors of forearm fair and equal on both sides. Dorsal flexion of the hand is possible to a greater extent in the left hand and the muscular strength there is somewhat better than in right. For the palmar flexors the same condition holds good. Grip on the left side is good, on the right subnormal. In the legs there is no apparent wasting, the right leg seems a little weaker than the left.

Reflexes.—Plantar not obtained on either side. Abdominal not obtained. Cremaster obtained on both sides; they are equal. Patellar reflexes are active on both sides, on the right exaggerated. Tendo Achillis reflex present on both sides, plus on the right. There is the most exquisite ankle-clonus on the right side, none on the left. Triceps: reflex well marked on the left, not obtained on the right. Periosteal reflex at wrist better marked on the left side.

Mechanical stimulation.—About the same on both sides.

Electrical Examination.

Faradic.

	LEFT.	RIGHT.
Nervus ulnaris	28	28
Nervus radialis	22–28	22–28
Nervus medianus	22	17–22
M. Biceps	17–22	13
" Triceps	22–28	22–28
" Deltoideus	22	22–28
" Extensor communis	22	28
" Extensor radialis	17–22	28
" Extensor ulnaris	17	22
" Extensor pollicis et indicis	22–28	28
" Extensor minimi digiti	28	—
" Supinator longus	22–28	22–28
" Flexor profundus	17–22	28–34
" Flexor ulnaris	28	28
" Flexor sublimus	28	28–34
" Pronator rad. ter	28	28–34
" Adductor pollicis	28	44–49
" Opponens pollicis	28–34	22–28
" Thenar	28	28–34
" Interossei	28	34
" Flexor minimi digiti	22	
" Adductor minimi digiti	28	

As there was really no difference between the two sides as regards the strength of the current which produced the smallest contraction, we concluded to stimulate all the muscles with a fairly strong faradic current, and note the difference in the strength of the contractions, which in some of the muscles was quite apparent. The result was as follows: With a current of 49, bicipites react equally. The reaction of the ulnar nerve is less strong and less quick and less tetanic on the right side; extensor communis less active on right side; supinatores almost equal; extensores radial. equal; extensores ulnares almost equal; pronatores equal; flexor. profund., flexor. carpi radial., flexor. sublim. equal on both sides; flexor carpi ulnaris perhaps a little more sluggish on the right side; the triceps more sluggish and less tetanic on right side; pectoralis less on right side; opponens and adductor pollicis decidedly less on right side.

Galvanic Current.

Left Arm.—

Ulnar nerve, $2\frac{1}{2}$ M. A., K. C. C. $<$ 6 M. A., A. C. C., A. O. C., 8.
Radial nerve, 2 M. A., K. C. C. $<$ $3\frac{1}{2}$ M. A., A. O. C., A. C. C., + 8.
Median nerve, $1\frac{1}{2}$ M. A., K. C. C. $<$ 3 M. A., A. O. C., A. C. C., + 8.
M. Deltoid, ant. part, 4 M. A., K. C. C. $>$ $3\frac{1}{2}$ M. A., A. C. C.
mid. part, $5\frac{1}{2}$ M. A., K. C. C. $>$ 5 M. A., A. C. C.
post. part, 6 M. A., K. C. C. $>$ $5\frac{1}{2}$ M. A., A. C. C. } quick.
" Triceps, $2\frac{3}{4}$ M. A., K. C. C. $<$ 5 M. A., A. C. C.
" Biceps, $1\frac{1}{2}$ M. A., K. C. C. $<$ $1\frac{3}{4}$ M. A., A. C. C.
" Supinator, 2 M. A., K. C. C. $<$ 3 M. A., A. C. C.
" Extensor radialis, $2\frac{1}{2}$ M. A., K. C. C. $<$ $4\frac{1}{2}$ M. A., A. C. C.
" Extensor ulnaris, $2\frac{1}{2}$ M. A., K. C. C. $<$ 4 M. A., A. C. C.
" Extensor communis, 4 M. A., K. C. C. — 4 M. A., A. C. C.
" Extensor pollicis, $4\frac{1}{2}$ M. A., K. C. C. $<$ 6 M. A., A. C. C. and A. O. C.
" Extensor indicis, 2 M. A., K. C. C. $<$ $2\frac{1}{2}$ M. A., A. C. C.
" Interossei, 2 M. A., K. C. C. $<$ $3\frac{1}{2}$ M. A., A. C. C.
" Thenar, $4\frac{1}{2}$ M. A., K. C. C. $>$ $3\frac{1}{2}$ M. A., A. C. C.
" Flexor carpi ulnaris, $2\frac{1}{2}$ M. A., K. C. C. $<$ 5 M. A., A. C. C.
" Flexor profundus, $2\frac{1}{2}$ M. A., K. C. C. $<$ $3\frac{1}{2}$ M. A., A. C. C.
" Flexor sublimis (II, III), 2 M. A., K. C. C. $<$ 5 M. A., A. C. C.
" Flexor sublimis (ind. and little), 2 M. A., K. C. C. $<$ 5 M. A., A. C. C.
" Adductor pollicis, 2 M. A., K. C. C. $<$ 6 M. A., A. C. C.
" Opponens, 3 M. A., K. C. C. $<$ 6 M. A., A. C. C.
" Flexor carpi radialis, 3 M. A., K. C. C. $<$ 5 M. A., A. C. C. All the contractions are quick, not sluggish.

Right Arm.—

Ulnar nerve, $1\frac{1}{2}$ M. A., K. C. C. < 5 M. A., A. C. C.
Radial nerve, $2\frac{1}{2}$ M. A., K. C. C. < $4\frac{1}{2}$ M. A., A. C. C.
Median nerve, $2\frac{1}{2}$ M. A., K. C. C. < $2\frac{3}{4}$ M. A., A. O. C., $8\frac{1}{2}$ M. A., A. C. C.
M. Extensor radialis, $3\frac{1}{2}$ M. A., K. C. C. < $5\frac{1}{2}$ M. A., A. C. C.
" Supinator, 3 M. A., K. C. C. — 3 M. A., A. C. C.
" Extensor ulnaris, 2 M. A., K. C. C. < 5 M. A., A. C. C.
" Extensor communis, 3 M. A., K. C. C. — 3 M. A., A. C. C.
" Extensor indicis, $2\frac{1}{2}$ M. A., K. C. C. < $4\frac{1}{2}$ M. A., A. C. C.
" Extensor pollicis, $2\frac{1}{2}$ M. A., K. C. C. < $4\frac{1}{2}$ M. A., A. C. C.
" Deltoideus, 2–$3\frac{1}{2}$ M. A., K. C. C. < $4\frac{1}{2}$–6 M. A., A. C. C.
" Flexor ulnar, $2\frac{1}{2}$ M. A., K. C. C. < A. C. C., not obtained.
" Pronator, 2 M. A., K. C. C. < 6 M. A., A. C. C.
" Flexor profundus, 2 M. A., K. C. C. < 4 M. A., A. C. C.
" Flexor sublimis, $2\frac{1}{2}$ M. A., K. C. C. < 6 M. A., A. C. C.
" Flexor pollicis, 3 M. A., K. C. C. < 6 M. A., A. C. C.
" Opponens, $3\frac{1}{2}$ M. A., K. C. C. < 5 M. A., A. C. C.
" Interossei, 3 M. A., K. C. C. < 4 M. A., A. C. C., slow.
" Thenar, $3\frac{1}{2}$ M. A., K. C. C. < 6 M. A., A. C. C., slow.
" Adductor pollicis, $5\frac{1}{2}$ M. A., K. C. C. < 8 + M. A., A. C. C., slow.

Sensation.—As the patient himself had noticed, there was a distinct difference in pain sensation on the side not affected by the motor paralysis. On the whole leg analgesia existed, and a pin was only felt when it penetrated quite deeply into the skin ; this involved half of the scrotum and half of the penis. On the body this was less noticeable, and was more marked in the lower parts and closer to the spinal column. Upwards this dulling of the sensation to pain extended to about the third rib in front, and about the same height on the back and down the back of the arm. It reached the median line anteriorly and posteriorly.

From about the middle of January there was no perceptible difference on the two arms. Over the same area and about similarly distributed as regards intensity, there was loss of temperature sense, this being abolished even to very marked differences of hot and cold, becoming less dull, however, in about the same area as the pain sensation did. *As to touch there was not the slightest dulling anywhere on the body.* A zone of hyperæsthesia could not be made out at any time while the patient was under observation, neither could any change in the muscular sense be determined.

To sum up then, *the patient fell from a train, sustained several injuries, but no perceptible one to the spine. No trace of paralysis for*

six days; at that time some pain in right extremities, and after some hours' sleep complete paralysis in the right leg and arm. On examination, three months after, weakness of triceps, pectoralis, flexors and extensors and small muscles of the hand of right arm, with decrease in electrical excitability in some of these muscles (triceps, pectoralis, some of the small muscles, extensor communis); also some visible atrophy; furthermore, stiffness in the right leg; no atrophy or electrical changes in muscles of leg; loss of the sense of temperature and pain on the whole non-paralysed left side.

We have here, therefore, a case of Brown-Sequard's paralysis, resembling the picture of a syringomeyelia.

The diseases which we have to think of in our differential diagnosis we shall consider now. We can readily in both cases exclude a cerebral lesion, but the spinal affections other than haematomyelia which can give rise to such an acute paralysis are: 1. Hemorrhage into the spinal canal outside of the cord or haematorrhachis. 2. Acute myelitis. 3. Acute ischaemic myelomalacia.

In Case II, it is not difficult to exclude haematorrhachis, for we cannot imagine that by such a hemorrhage sensation alone on one side and motion alone on the other should be affected. In the first case haematorrhachis might have suggested itself at the beginning, but the course which the disease took, and the nutritive disturbances in the muscles can hardly be brought into accord with that diagnosis, even if we assume the possibility of a clot forming between the dura and the bony canal; in that case we would also rather expect to find changes in the other arm as well. Besides the examples of such a localized clot are very few.[1]

Myelitis.—The strongest point against myelitis in our cases, especially Case I, is certainly the very acute onset. A paralysis which, without any prodromes save an acute pain lasting for a short time, and transient contractures, destroys *within a few minutes* motion and sensation from the arms down, cannot be explained in any other way than by hemorrhage. In Case II, while the onset was certainly very acute, we cannot say positively how long it took from the first symptoms to the complete paralysis; but the paralysis did not spread

[1] Moulton (Thèse de Strassbourg, 1887) quotes a case of this nature, reported by Laennec.

after the patient had awakened. An acute anterior polio-myelitis, of which we certainly have to think in Case I, while often very acute, never begins and reaches its highest development in so short a time. An anterior polio-myelitis is furthermore never accompanied with sensory changes, which have been quite marked in our case, and lastly it is accompanied by fever and general disturbances.

The quick regression of some of the symptoms which we found in Case I were also much in favor of hemorrhage. Symptoms of paralysis which disappear in such a short time cannot be due to inflammation and we have to look for another cause, and pressure naturally suggests itself. Krafft-Ebing,[1] in a somewhat similar case, in which, however, the hemorrhage was into the posterior horn, explained the motor paralysis by shock. The term "shock" does not convey any definite idea however, and in fact neither does pressure, but the symptoms seem to be to some extent explained by what is usually called pressure. No doubt infiltration and secondary inflammation play some part. Anterior polio-myelitis also may produce symptoms which by some are supposed to be due to pressure, owing to extravasation of blood. We would expect them in this disease, however, to come on later and to be less extensive.

Therefore, we can almost with certainty exclude myelitis in Case I. While we cannot, it is true, be so sure in Case II, still I think that with the acute onset the limitation of the apparent destruction is in favor of hemorrhage.

Here I may mention the three well-known cases described as unilateral hemorrhage, with symptoms of Brown-Séquard's paralysis, reported by Monod, Oré, Brechet.[2] Also a case which more closely resembles our Case II. It is one reported by Rosenbach and Schtscherbach,[3] under the name of syringomyelia. It is true the case resembles very much the complex of symptoms presented by syringomyelia, as in fact our case does, but considering that the patient had

[1] *Loc. cit.*

[2] Extracts of all three cases are given by Levier, *loc. cit.*; all three typical cases of Brown-Séquard's paralysis.

[3] Zur Casuistik der Syringomyelie, by Rosenbach and Schtscherbach, in St. Petersburg. Neurolog. Centralblatt (Mendel), 15 April, 1890, p. 226. Patient fell down several steps on his back; could not raise right foot; the hand had lost the motion of the fingers; gradual improvement. Half a year later atrophy of some muscles of arm; on unparalyzed side analgesia and loss of temperature sense.

had a fall, and immediately after it the symptoms had developed, it seems more natural to attribute these symptoms to a hemorrhage, just as in our patient, than to an acute exacerbation of a previously existing process which had not been noticed by the patient.

Acute ischaemic myelomalacia is one of the rarest affections and little is known about it. Leyden is said to have reported two cases in which he had found emboli in spinal arteries in the course of an acute ulcerative endocarditis. We can imagine how an embolism or thrombosis can give rise to some localized nutritive disturbances, such as Klebs has lately described,[1] but not to such a limited acute softening, for we have in the cord a different vascular condition from the brain and not a system of end-arteries. But even if this could happen, the softening ought to be confined to the area in which destruction remained, and we would have no explanation for the extensive pressure symptoms in Case I. Cases have been reported in which complete myelomalacia followed cutting off of the blood supply in the abdominal aorta.

We have yet to consider the influence which the accident had in the production of the hemorrhage in these two cases. It seems hardly conceivable that in both the concussion which preceded—even though there intervened a considerable time between it and the spinal symptoms—should be in no causal relation with the disease. Far from endeavoring to give a definite explanation of this *nexus causalis*, which would in fact be impossible, I think it will be interesting to consider the spinal lesions produced by a concussion. It is not uncommon to find a hemorrhage following immediately either a fall or blow on the back. Such cases have often been described, and we find some good examples in the above-mentioned work of Thorburn; but the changes seem more frequently to be of a different nature. Symptoms of a subacute myelitis, or it has been claimed, of a distinct system-disease (tabes), may appear, or a paralysis occurs which is not explained by any appreciable anatomical changes. What are the early changes which are produced by concussion? Schmaus,[2] in endeavoring to answer this question has studied it experimentally and

[1] Klebs: Ueber Landry'sche Paralyse. Deutsche Medicin. Wochenschrift, 15 Januar. 1891.

[2] Dr. Hans Schmaus: Beiträge zur pathologischen Anatomie der Rückenmarkserschütterung. Virchow's Archiv, Band 122, pp. 328 and 470.

shown that the first anatomically appreciable changes consist in death of the axis-cylinders. In the anterior horns was found an indefinite granular degeneration. Hemorrhage and changes in the vessels did not occur, but he observed circumscribed foci of softening with cavity formation, and also gliosis with cavity formation. (Each of these changes in one experiment; on the animal with extensive gliosis the autopsy was made twelve days after onset of disease).

There is still another class of which Gowers speaks, where we have no symptoms immediately after the concussion, but where later signs of subacute myelitis appear. The last class then would be formed by cases such as ours,[1] in which there are no symptoms for a longer or shorter period of time, and then a sudden onset. The cases are undoubtedly rare, and it is difficult to interpret them. Had any of the above-mentioned changes taken place, we would have expected them to be manifested by symptoms. That we find gliosis produced by concussion cannot explain Case II—although it resembles the clinical picture of syringomyelia—for the same reason. Whether we then have a change produced chiefly in the vessels without any parenchymatous alterations sufficient to cause symptoms can only be a supposition. This delay in the effect of the concussion is certainly not the least interesting feature of our cases.

[1] There are two cases reported under the title of Haematomyelia, which are of interest in this connection. The case of Peddie (Monthly Journal of Medical Science. of July, 1846, and June, 1847, pp. 819–833), in which ten days after spinal concussion an acute paralysis came on. The case of McMunn (Dublin Journal of Medical Science, Vol. 59, 1880, pp. 182–189), in which two days after spinal concussion the acute paralysis came on. In both cases, no symptoms in the intervening time.